Dietary Diabetes Risk Reduction Score, Race and Ethnicity, and Risk of Type 2 Diabetes in Women

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EPIDEMIOLOGY/HEALTH SERVICES RESEARCH

OBJECTIVE

To evaluate racial and ethnic differences in the association between a dietary diabetes risk reduction score and incidence of type 2 diabetes in U.S. white and minority women.

RESEARCH DESIGN AND METHODS

We followed 156,030 non-Hispanic white (NHW), 2,026 Asian, 2,053 Hispanic, and 2,307 black women in the Nurses' Health Study (NHS) (1980–2008) and NHS II (1991–2009). A time-updated dietary diabetes risk reduction score (range 8–32) was created by adding points corresponding with each quartile of intake of eight dietary factors (1 = highest risk; 4 = lowest risk). A higher score indicates a healthier overall diet.

RESULTS

We documented 10,922 incident type 2 diabetes cases in NHW, 157 in Asian, 193 in Hispanic, and 307 in black women. Multivariable-adjusted pooled hazard ratio across two cohorts for a 10th–90th percentile range difference in dietary diabetes risk reduction score was 0.49 (95% CI 0.46, 0.52) for NHW, 0.53 (0.31, 0.92) for Asian, 0.45 (0.29, 0.70) for Hispanic, 0.68 (0.47, 0.98) for black, and 0.58 (0.46, 0.74) for overall minority women (*P* for interaction between minority race/ethnicity and dietary score = 0.08). The absolute risk difference (cases per 1,000 person-years) for the same contrast in dietary score was −5.3 (−7.8, −2.7) for NHW, −7.2 (−22.9, 8.4) for Asian, −11.6 (−26.7, 3.5) for Hispanic, −6.8 (−19.5, 5.9) for black, and −8.0 (−15.6, −0.5) for overall minority women (*P* for interaction = 0.04).

CONCLUSIONS

A higher dietary diabetes risk reduction score was inversely associated with risk of type 2 diabetes in all racial and ethnic groups, but the absolute risk difference was greater in minority women.

According to the American Diabetes Association, approximately 29.1 million people or 9.1% of the U.S. population currently has diabetes (1). Overall, the prevalence of diabetes has been increasing at an alarming rate in the U.S and worldwide (2), and there is strong evidence for racial and ethnic differences in the prevalence of metabolic syndrome and type 2 diabetes (3,4). In an earlier report from the Nurses' Health Study (NHS), Shai et al. (5) found a significantly higher risk for type 2 diabetes in Asians, Hispanics, and blacks, compared with whites, after accounting for BMI or overall adiposity and behavioral risk factors.

Previous studies have reported that various dietary factors are associated with the risk of type 2 diabetes. Cereal fiber (6), nut (7), and coffee (8) consumption and

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higher ratio of polyunsaturated to saturated fats (P:S) (9) are inversely associated with the risk of type 2 diabetes, whereas glycemic index (GI) (10–12) and *trans* fat (9), sugar-sweetened beverage (SSB) (13,14), and red meat and other processed meat (15,16) consumption are positively associated with type 2 diabetes risk. Most of the studies that support these findings, however, have been conducted in predominantly white populations (6–12,14–16).

In the current study, we examined whether the dietary determinants of type 2 diabetes observed in predominantly white groups were similar to those in other racial and ethnic groups. We created a dietary diabetes risk reduction score that included components more recently found to be associated with risk of type 2 diabetes, including SSBs, coffee, nuts, and red and processed meats, in addition to the four components used in an earlier analysis, which included GI, cereal fiber, P:S, and *trans* fat (5). With longer follow-up in NHS (5) and the inclusion of women in NHS II, we were able to conduct analyses within specific racial and ethnic groups. We also examined the association between individual components of the dietary diabetes risk reduction score and risk of type 2 diabetes in non-Hispanic white (NHW) and minority (consisting of Asian, Hispanic, and black) women.

RESEARCH DESIGN AND METHODS

Study Population

The NHS is a prospective study of 121,700 registered female nurses who were aged 30–55 years at the time of enrollment in 1976. With a similar study design, NHS II includes 116,430 younger female nurses aged 25–42 years when enrolled in 1989. Participants provided information on their medical history and other lifestyle and health-related risk factors for cancer and cardiovascular disease on the baseline questionnaire. Subsequently, biennial questionnaires were used to update this information and identify new health outcomes. In NHS, diet was first assessed in 1980 using a food frequency questionnaire (FFQ) and updated approximately every 4 years thereafter, whereas in NHS II, diet was first assessed in 1991 and updated every 4 years since then.

In this study, we excluded women with a prior diagnosis of diabetes, cancer, or cardiovascular disease, as reported on the 1980 and 1991 or earlier questionnaires from NHS and NHS II, respectively (n = 7,699 in NHS and n = 7,423 in NHS II). Additionally, we excluded women who did not return diet questionnaires, left >70 food items blank on the baseline FFQ, or reported implausible levels of total energy intake (<500 kcal/day or >3,500 kcal/day) (n = 29,293 in NHS and n = 21,191 in NHS II). Women who did not provide baseline data on dietary factors of interest were excluded (n = 1,633 in NHS and n = 338 in NHS II). Participants reported their race and ethnicity in 1992 and 2004 in NHS and in 1989 and 2005 in NHS II as NHW (southern European/Mediterranean, Scandinavian, and other Caucasian ancestry), black, Asian, American Indian, Hawaiian, or Hispanic. Participants of American Indian or Hawaiian background were excluded owing to their small sample size (<1% of the study population). Lastly, we excluded participants without follow-up information on diabetes diagnosis date (n = 2,181 in NHS and n = 553 in NHS II). We combined Asian, Hispanic, and black women into a category called the overall minority group. After these exclusions, we had data available for 156,030 NHW, 2,026 Asian, 2,053 Hispanic, and 2,307 black women for the final analysis. This study was approved by the Institutional Review Board, the Brigham and Women's Hospital, and/or the Harvard School of Public Health.

Dietary Assessment

Diet was assessed using validated semiquantitative FFQs in 1980. Participants were asked to specify, on average, how often they consumed each food as indicated by the unit or portion size on the questionnaire during the previous year. The frequencies of consumption were listed in a multiple-choice fashion as follows: almost never, one to three times per month, once per week, two to four times per week, five to six times per week, once per day, two to three times per day, four to six times per day, or more than six times per day. Nutrient intakes were then calculated by multiplying the frequency of consumption of a specified unit or portion size of food by its nutrient content (17). The FFQ has been shown to perform well in NHS, and data on reproducibility and validity in this cohort, as well as detailed descriptions of both abbreviated and expanded FFQ forms and nutrient intake calculation procedures, have been previously documented (18,19). A validation study conducted among a subset of women in NHS also reported relatively high correlation coefficients between the FFQ and multiple dietary records for carbohydrates (0.64) (12), fiber (0.56) (12), nuts and peanut butter (0.75) (7), SSBs (0.84 for sugar-sweetened and diet sodas, 0.56 for other carbonated soft drinks, and 0.56 for fruit punch) (20), coffee (0.78) (21), total and specific types of fat (0.46–0.68) (9), and red and processed meats (0.56 for hot dogs, 0.70 for bacon, 0.55 for other processed meats, 0.38 for hamburgers, and 0.46 for red meat) (16).

We calculated time-updated cumulative averages of dietary intake data from baseline to censoring events using repeated FFQs. Using a cumulative average of repeated dietary measures can be useful because it can reduce random within-person error and better assess true long-term diet (22). We stopped updating dietary variables when the participants reported a diagnosis of stroke, myocardial infarction, angina, or cancer, since these conditions could lead to changes in dietary intakes.

Computation of Dietary Diabetes Risk Reduction Score

We assigned each woman a score between one (highest risk quartile) and four (lowest risk quartile) that corresponded to her quartile of intake for the following dietary factors: cereal fiber, nuts, coffee, and P:S in ascending order and GI, *trans* fat, SSBs, and red and processed meats in descending order. The dietary diabetes risk reduction score was calculated as the sum of these quartile values with a range of 8–32, and a higher score indicates a healthier overall diet.

Follow-up and Ascertainment of Incident Cases of Type 2 Diabetes

The primary outcome of interest was incidence of type 2 diabetes. We used data from biennial follow-up questionnaires (starting in 1982 for NHS and 1993 for NHS II) on which women were asked whether diabetes had been newly diagnosed. Additionally, participants were asked to complete a supplementary questionnaire to confirm the

diagnosis and provide various details such as the date of diagnosis, types of diagnostic tests used, symptoms, and medications prescribed once a new case of diabetes was reported. In accordance with the criteria proposed by the American Diabetes Association, the diagnosis of type 2 diabetes was established if at least one of the following was reported on the supplemental questionnaire according to the 1997 American Diabetes Association criteria: *1*) one or more classic symptoms (excessive thirst, polyuria, weight loss, hunger) plus fasting plasma glucose concentrations of at least 126 mg/dL (7.0 mmol/L) or random plasma glucose concentrations of at least 200 mg/dL (11.1 mmol/L), *2*) at least two elevated plasma glucose concentrations on different occasions (fasting concentrations of at least 126 mg/dL [7.0 mmol/L], random plasma glucose concentrations of at least 200 mg/dL [11.1 mmol/L], and/or concentrations of at least 200 mg/dL [11.1 mmol/L] after $\geq$2 h shown by oral glucose tolerance testing) in the absence of symptoms, or *3*) treatment with hypoglycemic medication (insulin or oral hypoglycemic agent).

The high validity of self-reported cases of diabetes in NHS has been previously reported, and the diagnosis corresponded with medical records in 98% of the cases (23). We expect the results to be similar in NHS II.

Assessment of Covariates and Nondietary Factors

We used information from the baseline and biennial follow-up questionnaires to include the following covariates in the analysis: age, physical activity, smoking, family history of diabetes, alcohol intake, postmenopausal status and menopausal hormone use, oral contraceptive use (NHS II only), total energy intake, and BMI. Women provided information on their weight and smoking status on the baseline questionnaires, and this information was updated every 2 years. Height was assessed at baseline only. In NHS, self-reported body weight has been reported to be highly correlated with weight measured by a technician (r = 0.96) (24).

Statistical Analysis

We assessed the association between time-updated dietary diabetes risk reduction score and incidence of type 2 diabetes by using a Cox proportional hazards regression model. We estimated hazard ratios (HRs) of type 2 diabetes and 95% CI for each quartile of dietary diabetes risk reduction score compared with the lowest quartile (reference category). We calculated each individual's person-years from the date of return of the baseline questionnaire to the date of diagnosis of type 2 diabetes, death, or the end of follow-up (30 June 2008 for NHS and 30 June 2009 for NHS II)—whichever came first.

In the multivariate analysis, we simultaneously controlled for age and calendar time with updated information at each 2-year questionnaire cycle, including smoking status (never or ever), alcohol intake (g/day), physical activity (3, 3–8.9, 9–17.9, 18–26.9, or $\geq$27 MET h/week), family history of diabetes, postmenopausal status and menopausal hormone use (never, past, or current), oral contraceptive use (NHS II only) (never, past, or current), and total energy intake. Since BMI can act as an intermediate variable between diet and diabetes, we included BMI in a separate model as a time-varying covariate. We created a modified dietary diabetes risk reduction score when examining the association between individual components of the dietary score and type 2 diabetes risk by excluding the component being considered from the total dietary diabetes risk reduction score and adjusting for this modified score in the multivariate model. We tested for linear trend by assigning the median value to each quartile and modeling it as a continuous variable. We also computed HRs for a 10th–90th percentile range difference in dietary diabetes risk reduction score, GI, and P:S; a 1% increase (% calories) in *trans* fat intake; and an increase of 1 g/day of cereal fiber intake; of 1 serving/day of SSB, nuts, and red and processed meats; and of 1 cup/day of coffee. Dietary missing values during follow-up were replaced with cumulative means by the carry-forward method. Nondietary missing values were replaced with the last value carried forward from a previous 2-year cycle. If the value at the previous 2-year cycle was missing, a missing value indicator was created. After conducting separate analyses in NHS and NHS II to estimate study-specific HRs, we computed pooled summary estimates across the two studies using a fixed-effects model in which the studies were weighted proportionately to the inverse of the study-specific variance.

To test for multiplicative interaction between race/ethnicity and diet, we added the interaction term (minority race/ethnicity $\times$ dietary diabetes risk reduction score) along with the terms minority race/ethnicity (dichotomous) and dietary diabetes risk reduction score in the regression model; exposure distributions in the overall population were used to define the 10th and 90th percentiles. We also calculated multivariate-adjusted differences in absolute risks by performing a log-binomial regression analysis and tested for additive interaction using interaction terms (25,26). All statistical analyses were performed using SAS software package, version 9.2 (SAS Institute).

RESULTS

We documented 10,922 incident type 2 diabetes cases in NHW (7,322 in NHS and 3,600 in NHS II), 157 in Asian (84 in NHS and 73 in NHS II), 193 in Hispanic (87 in NHS and 106 in NHS II), and 307 in black (177 in NHS and 130 in NHS II) women during a maximum of 28 years of follow-up in NHS and a maximum of 18 years in NHS II. Table 1 displays the distribution of age-adjusted baseline characteristics of women in NHS and NHS II by race and ethnicity. Compared with NHW women, Asian women had lower mean BMI and alcohol consumption but higher mean dietary GI, P:S, and intakes of SSBs and nuts. Asian women also consumed less *trans* fat and coffee, on average, compared with NHW women but had higher mean total energy intake. Similar patterns were observed in Hispanic women, though Hispanic women were more physically active and had higher intake of cereal fiber and lower intake of red and processed meats, on average, compared with NHW women. Total energy intake did not differ much between the two groups. In general, black women were more likely to have higher mean BMI and be less physically active compared with NHW women. They also had higher mean GI, P:S, and intakes of SSBs and nuts but lower intakes of cereal fiber, *trans* fat, and coffee compared with NHW women. A higher percentage of minority women had a family history of diabetes compared with NHW

Table 1—Baseline age-standardized characteristics of women without type 2 diabetes across racial/ethnic groups in the NHS and NHS II*

	Racial and ethnic groups			
	White†	Asian	Hispanic	Black
N	156,030	2,026	2,053	2,307
Age (years)‡	40.4 ± 7.6	39.0 ± 6.8	38.6 ± 6.9	40.5 ± 7.0
BMI (kg/m^2)	24.4 ± 4.8	22.6 ± 3.5#	24.8 ± 4.7#	26.5 ± 5.5#
Family history of diabetes (%)	31.0	40.2#	43.2#	44.8#
Physical activity (MET h/week)	17.1 ± 24.1	17.4 ± 27.1	19.7 ± 30.8#	16.1 ± 24.8#
Current smoker (%)	19.0	8.1#	11.4#	18.1
Hypertension (%)	9.5	7.7#	7.5#	20.5#
Hypercholesterolemia (%)	9.9	13.8#	13.5#	14.2#
Postmenopausal (%)	21.9	15.6#	17.1#	20.8#
Alcohol intake (g/day)	4.6 ± 8.4	1.6 ± 5.5#	3.6 ± 7.5#	2.9 ± 6.7#
GI	52.8 ± 4.0	55.2 ± 4.3#	53.0 ± 4.0#	53.8 ± 4.5#
Cereal fiber (g/day)	4.3 ± 2.9	4.0 ± 2.4	4.6 ± 3.1#	3.6 ± 2.6#
Polyunsaturated fat (% energy)	5.5 ± 1.5	5.2 ± 1.4#	5.3 ± 1.4#	5.4 ± 1.5#
Saturated fat (% energy)	13.2 ± 3.7	11.2 ± 3.7#	12.1 ± 3.7#	12.0 ± 3.5#
P:S	0.45 ± 0.17	0.52 ± 0.21#	0.48 ± 0.18#	0.49 ± 0.20#
Trans fat (% energy)	1.9 ± 0.7	1.6 ± 0.7#	1.7 ± 0.7#	1.8 ± 0.7#
SSB intake (servings/day)	0.39 ± 0.75	0.52 ± 0.79#	0.45 ± 0.85#	0.68 ± 0.99#
Coffee intake (cups/day)	1.9 ± 1.8	1.3 ± 1.5#	1.5 ± 1.5#	1.1 ± 1.4#
Nut intake (servings/day)	0.10 ± 0.22	0.12 ± 0.24#	0.09 ± 0.19#	0.13 ± 0.28#
Red and processed meat intake (servings/day)	1.2 ± 0.9	1.2 ± 1.0	1.1 ± 0.9#	1.2 ± 1.0
Total energy intake (kcal/day)	1,690 ± 534	1,733 ± 602#	1,707 ± 585	1,676 ± 604#
Dietary diabetes risk reduction score¶	20.3 ± 4.0	19.8 ± 3.8#	20.5 ± 4.0	19.3 ± 4.1#

*Values are standardized to the age distribution of the study population and represent means ± SD except where % are shown. †Includes non-Hispanic women with southern European/Mediterranean ancestry, Scandinavian ancestry, and other Caucasian ancestry. ‡Value is not age adjusted. ¶Sum of quartile values (1–4) for intakes of cereal fiber, P:S, coffee, and nuts (ascending order) and GI, *trans* fat, SSBs, and red and processed meat (descending order); higher score indicates a healthier overall diet. #$P < 0.05$ compared with whites.

women, and mean alcohol intake was lower among minorities than among NHWs. Compared with NHW women, the mean dietary diabetes risk reduction score was slightly lower among Asian and black women, but the quantitative differences were small.

The pooled HRs of type 2 diabetes risk according to quartile of dietary diabetes risk score for each racial and ethnic group are shown in Table 2. In age- and multivariate-adjusted models, dietary diabetes risk reduction score was inversely associated with the risk of development of type 2 diabetes in all racial and ethnic groups (all *P* for trend$<$0.05) as well as in overall minority women (*P* for trend$<$0.001). Further adjustment for BMI attenuated some of these associations, particularly in NHW and Hispanic women. In the main multivariate analysis that did not adjust for BMI, within each racial and ethnic group, women in the highest quartile of the dietary diabetes reduction risk score had a lower risk of type 2 diabetes compared with those in the lowest quartile: NHW (HR 0.52 [95% CI 0.49, 0.56]), Asian (0.58 [0.35, 0.95]), Hispanic (0.45 [0.28, 0.74]), black (0.68 [0.48, 0.97]), and overall minority (0.64 [0.51, 0.80]).

After adjustment for age and other confounders, HR for a 10th–90th percentile range difference in dietary diabetes reduction risk score and type 2 diabetes was as follows: 0.49 (95% CI 0.46, 0.52) in NHW, 0.53 (0.31, 0.92) in Asian, 0.45 (0.29, 0.70) in Hispanic, 0.68 (0.47, 0.98) in black, and 0.58 (0.46, 0.74) in overall minority women. The *P* value for multiplicative interaction between dietary diabetes risk reduction score and minority race/ethnicity was 0.08.

The pooled estimates of HRs and 95% CI of type 2 diabetes risk according to intakes of individual components of the dietary diabetes risk reduction score in NHW and overall minority women are shown in Table 3. In NHW women, intakes of GI, *trans* fat, SSBs, and red and processed meats were positively associated with risk of type 2 diabetes (all $P <$ 0.05), and intakes of cereal fiber, coffee, and nuts were inversely associated with risk of type 2 diabetes (all $P < 0.05$). All of these associations were similar in magnitude in overall minority women, but only GI (positively), intakes of cereal fiber (inversely), SSBs (positively), coffee (inversely), and red and processed meats (positively) were significantly associated with type 2 diabetes risk. The 95% CI for all components in overall minority women included HR observed in NHW women.

The absolute risk difference is expected to be greater for high-risk than for low-risk populations. The absolute risk difference in cases per 1,000 person-years for the 10th–90th percentile range difference in dietary diabetes risk reduction score was −5.3 (−7.8, −2.7) for NHW, −7.2 (−22.9, 8.4) for Asian, −11.6 (−26.7, 3.5) for Hispanic, −6.8 (−19.5, 5.9) for black, and −8.0 (−15.6, −0.5) for overall minority women. The *P* value for interaction between dietary diabetes risk reduction score and minority race/ethnicity on the absolute scale was 0.04.

Table 2—Pooled estimates of HRs (95% CI) of type 2 diabetes risk according to quartile of dietary diabetes risk reduction score across racial and ethnic groups in the NHS and NHS II*

	Quartile of dietary diabetes risk reduction score					
	Quartile 1	Quartile 2	Quartile 3	Quartile 4	P_{trend}*	HR (95% CI) for 10th–90th percentile range difference
White†						
Median (IQ range)						
NHS	15 (14–16)	19 (18–19)	21 (21–22)	25 (24–27)		
NHS II	15 (13–16)	18 (18–19)	21 (21–22)	25 (24–27)		
Cases/person-years						
NHS	2,511/455,588	1,784/422,739	1,887/501,363	1,140/432,293		
NHS II	1,274/370,474	943/357,421	865/414,736	518/367,153		
Age-adjusted model	1.00	0.72 (0.69, 0.76)	0.58 (0.55, 0.61)	0.40 (0.38, 0.43)	<0.001	0.36 (0.34, 0.38)
Multivariate model 1§	1.00	0.79 (0.75, 0.83)	0.69 (0.65, 0.72)	0.52 (0.49, 0.56)	<0.001	0.49 (0.46, 0.52)
Multivariate model 2‡	1.00	0.85 (0.81, 0.89)	0.75 (0.71, 0.79)	0.61 (0.57, 0.65)	<0.001	0.57 (0.54, 0.61)
Asian						
Median (IQ range)						
NHS	15 (14–16)	18 (18–19)	21 (20–22)	24 (23–26)		
NHS II	15 (14–16)	18 (17–19)	21 (20–22)	24 (23–26)		
Cases/person-years						
NHS	23/3,182	21/3,793	24/3,873	16/3,379		
NHS II	25/6,414	23/6,587	11/6,568	14/5,854		
Age-adjusted model	1.00	0.74 (0.48, 1.12)	0.64 (0.40, 1.02)	0.57 (0.36, 0.92)	0.02	0.53 (0.31, 0.90)
Multivariate model 1§	1.00	0.80 (0.52, 1.24)	0.67 (0.42, 1.09)	0.58 (0.35, 0.95)	0.02	0.53 (0.31, 0.92)
Multivariate model 2‡	1.00	0.86 (0.55, 1.35)	0.70 (0.42, 1.14)	0.61 (0.36, 1.02)	0.04	0.56 (0.32, 0.97)
Hispanic						
Median (IQ range)						
NHS	15 (14–17)	19 (18–20)	22 (21–22)	25 (24–26)		
NHS II	15 (14–16)	19 (18–20)	22 (21–23)	26 (25–27)		
Cases/person-years						
NHS	33/3,765	25/3,283	21/3,763	8/3,589		
NHS II	31/6,306	28/6,557	26/6,229	21/6,478		
Age-adjusted model	1.00	0.76 (0.52, 1.11)	0.62 (0.42, 0.93)	0.39 (0.26, 0.60)	<0.001	0.39 (0.26, 0.60)
Multivariate model 1§	1.00	0.83 (0.56, 1.23)	0.69 (0.46, 1.04)	0.45 (0.28, 0.74)	<0.001	0.45 (0.29, 0.70)
Multivariate model 2‡	1.00	0.94 (0.62, 1.42)	0.77 (0.50, 1.18)	0.57 (0.35, 0.93)	0.01	0.55 (0.35, 0.85)
Black						
Median (IQ range)						
NHS	15 (14–16)	19 (18–19)	21 (21–22)	25 (24–27)		
NHS II	13 (12–15)	17 (16–18)	20 (19–21)	24 (23–25)		
Cases/person-years						
NHS	72/6,049	31/5,652	48/5,284	26/5,972		
NHS II	27/5,997	45/5,724	20/5,398	38/6,144		
Age-adjusted model	1.00	0.87 (0.63, 1.20)	0.82 (0.59, 1.13)	0.64 (0.45, 0.90)	0.01	0.62 (0.43, 0.89)
Multivariate model 1§	1.00	0.91 (0.66, 1.26)	0.84 (0.60, 1.17)	0.68 (0.48, 0.97)	0.04	0.68 (0.47, 0.98)
Multivariate model 2‡	1.00	0.85 (0.61, 1.19)	0.77 (0.55, 1.08)	0.71 (0.49, 1.01)	0.07	0.70 (0.48, 1.03)
Minorities¶						
Median (IQ range)						
NHS	15 (14–16)	19 (18–19)	21 (21–22)	25 (24–26)		
NHS II	14 (13–15)	18 (17–19)	21 (20–22)	25 (24–26)		
Cases/person-years						
NHS	128/12,996	77/12,729	93/12,921	50/12,939		
NHS II	83/18,717	96/18,868	57/18,195	73/18,476		
Age-adjusted model	1.00	0.81 (0.66, 1.00)	0.70 (0.56, 0.87)	0.54 (0.43, 0.68)	<0.001	0.53 (0.42, 0.67)
Multivariate model 1§	1.00	0.90 (0.73, 1.11)	0.75 (0.61, 0.93)	0.64 (0.51, 0.80)	<0.001	0.58 (0.46, 0.74)
Multivariate model 2‡	1.00	0.91 (0.74, 1.13)	0.75 (0.60, 0.93)	0.65 (0.49, 0.80)	<0.001	0.61 (0.48, 0.77)

IQ, interquartile. *P_{trend} was calculated by assigning median values to each quartile and was treated as a continuous variable. †Includes non-Hispanic women with southern European/Mediterranean ancestry, Scandinavian ancestry, and other Caucasian ancestry. §The multivariate model was adjusted for age, family history of diabetes, smoking status, physical activity level, alcohol intake, postmenopausal status and menopausal hormone use, oral contraceptive use (NHS II), and total energy intake. ‡The multivariate model was adjusted for variables in model 1 and BMI. ¶Includes Asian, Hispanic, and black women.

CONCLUSIONS

In these cohorts of women, we observed that a higher dietary diabetes risk reduction score was inversely associated with risk of type 2 diabetes in women of all individual racial and ethnic groups as well as in overall minority women. Although the relative risk estimates were similar across racial and ethnic groups, the absolute risk difference for the same contrast in dietary score was larger for the

Table 3—Pooled estimates of HRs (95% CI) of type 2 diabetes risk according to intake of individual components of the dietary diabetes risk reduction score for white and minority women in NHS and NHS II*†

	NHS	NHS II	Pooled
GI			
White‡	1.29 (1.19, 1.39)	1.06 (0.95, 1.18)	1.21 (1.13, 1.28)
Minorities§	1.47 (1.02, 2.11)	1.24 (0.85, 1.82)	1.36 (1.04, 1.77)
Cereal fiber (g/day)			
White	0.90 (0.88, 0.92)	0.91 (0.89, 0.93)	0.90 (0.89, 0.92)
Minorities	0.90 (0.83, 0.98)	0.98 (0.92, 1.03)	0.95 (0.90, 0.99)
P:S			
White	0.87 (0.81, 0.95)	1.09 (0.98, 1.21)	0.95 (0.89, 1.01)
Minorities	0.97 (0.65, 1.45)	0.92 (0.61, 1.37)	0.94 (0.71, 1.25)
Trans fat (% energy)			
White	1.01 (0.96, 1.06)	1.28 (1.18, 1.38)	1.08 (1.04, 1.13)
Minorities	1.00 (0.77, 1.30)	1.09 (0.84, 1.41)	1.04 (0.87, 1.25)
SSBs (servings/day)			
White	1.42 (1.28, 1.57)	1.15 (1.07, 1.25)	1.25 (1.17, 1.33)
Minorities	1.51 (1.01, 2.27)	1.01 (0.73, 1.24)	1.16 (1.08, 1.25)
Coffee (cups/day)			
White	0.92 (0.91, 0.93)	0.84 (0.81, 0.86)	0.90 (0.89, 0.91)
Minorities	0.91 (0.83, 0.98)	0.81 (0.72, 0.92)	0.88 (0.82, 0.94)
Nuts (servings/day)			
White	0.53 (0.42, 0.67)	0.78 (0.48, 1.20)	0.57 (0.47, 0.70)
Minorities	0.88 (0.33, 2.33)	0.32 (0.07, 1.43)	0.65 (0.29, 1.47)
Red and processed meats (servings/day)			
White	1.25 (1.19, 1.32)	1.63 (1.49, 1.77)	1.34 (1.28, 1.40)
Minorities	1.05 (0.85, 1.28)	1.31 (1.02, 1.69)	1.14 (1.02, 1.35)

*HRs (95% CI) for 10th–90th percentile range of GI and P:S, 1% increase (% calories) in *trans* fat intake, and an increase of 1 g/day of cereal fiber intake; 1 serving/day of SSBs, nuts, and red and processed meats; and 1 cup/day of coffee. †The multivariate model was adjusted for age, family history of diabetes, smoking status, physical activity level, alcohol intake, postmenopausal status and menopausal hormone use, oral contraceptive use (NHS II), total energy intake, and modified dietary diabetes risk reduction score. ‡Includes non-Hispanic women with southern European/Mediterranean ancestry, Scandinavian ancestry, and other Caucasian ancestry. §Includes Asian, Hispanic, and black women.

minority women compared with NHW women.

Although the importance of adopting a healthier lifestyle through modification of diet in diabetes prevention has been well established (27,28), there is limited evidence on whether these findings apply to racial and ethnic groups that are at elevated risk of diabetes. In epidemiological analyses, evidence on racial and ethnic differences in the relation between diet and risk of diabetes has been inconsistent (5,29–31). In this study, we found reduced risks of type 2 diabetes of similar magnitudes, with higher dietary diabetes risk reduction score representing healthier diet in all racial and ethnic groups. Overall, these findings are consistent with those observed by Qiao et al. (32) in a study that examined racial/ethnic differences in the association between dietary quality, as measured by baseline Alternate Healthy Eating Index (AHEI), and the risk of incident diabetes. They found that higher dietary quality predicted lower risk of diabetes in white and Hispanic women but not in black or Asian women. Compared with their findings, the associations we observed in the current study were generally stronger, possibly because we used updated dietary data and our dietary score reflected more recent findings for diet and diabetes risk. In addition, we found significant inverse associations in all racial and ethnic minority groups, whereas Qiao et al. observed wide CIs that precluded them from finding significant associations in all minority groups.

Our overall findings are consistent with those of previous observational studies that were of predominantly NHW women (5–12,14–16). Few studies have compared associations between specific dietary factors such as GI and cereal fiber and risk of type 2 diabetes across different racial and ethnic groups. In our study, we observed that higher GI and lower cereal fiber intake were associated with greater risk of type 2 diabetes in minority women. These results are consistent with those from a few studies of minority women (33,34), although in another study cereal fiber was inversely associated with risk of type 2 diabetes in whites, but the role of GI and dietary fiber in diabetes in blacks was unclear (35).

In previous prospective cohort studies, daily servings of SSB consumption have been reported to be associated with increased risk of type 2 diabetes in both NHW and black women (13,14,36). We found a 25% higher risk of developing type 2 diabetes with an increase of one serving of SSB per day in NHW women and 16% higher risk in minority women. The association between SSB and type 2 diabetes risk may be mediated by weight gain because SSBs are energy-dense beverages that may induce weight gain (37). Prior research has shown that SSBs generate low satiety and are often consumed in addition to usual food intake, leading to an increased energy intake and ultimately weight gain (38). When we adjusted for BMI in a separate model, the association remained positive and significant in both NHW and minority women (data not shown), suggesting that the association of SSBs with type 2 diabetes risk cannot be entirely attributed to BMI. In contrast to the potential harmful effects of SSBs on type 2 diabetes risk, there is strong epidemiologic evidence that supports a protective association of coffee with the risk of type 2 diabetes (8,21). In our study, we found a 10% reduction in risk of diabetes per one cup increase of coffee per day in NHW women and 12% reduction in risk in overall minority women.

There is a dearth of evidence on racial and ethnic differences in the association between red and processed meat intake and risk of type 2 diabetes, but one study found risks that were similar in magnitude in Caucasian women and Japanese American women (29). The findings from the current study indicate that higher intake of red and processed meats is positively associated with type 2 diabetes risk in both white (34% higher risk) and minority (14% higher risk) women. Furthermore, similar to the findings of Salmerón et al. (9), we found a significant positive association between *trans* fat intake and risk of type

2 diabetes in NHW women; however, the association was not significant in minority women. P:S was not significantly associated with type 2 diabetes risk in NHW or minority women. Further studies of intakes of different types of fats and red and processed meats and type 2 diabetes in different racial and ethnic populations are needed to confirm whether there are racial and ethnic differences in the association between these dietary factors and type 2 diabetes risk.

One notable finding of this study was that despite similar relative risk estimates across racial and ethnic groups, the absolute risk difference for the same contrast in dietary score was larger for minority women compared with white women. Minorities tend to have lower insulin sensitivity and insulin secretion capacity compared with NHWs, which could explain the high prevalence of insulin resistance and increased risk of diabetes in minority populations (4,5). Since minority populations are at higher absolute risk of type 2 diabetes, the benefit that can be conferred through a healthy diet may be greater in these populations than for NHW women, as suggested by our findings. Given expected mean differences in BMI across different racial and ethnic groups, we ran another analysis using waist circumference in a subset of participants (76%) who had this anthropometric data available. However, the magnitude of associations did not change appreciably by adjusting for waist circumference in place of BMI, although the CIs widened somewhat owing to smaller sample size (data not shown).

The strengths of our study include a large overall sample size, long follow-up, use of repeated and validated dietary questionnaires, updated computation of dietary diabetes risk reduction score, and high specificity of self-reported cases of type 2 diabetes as confirmed by a supplementary questionnaire. A limitation of our study is the modest sample size of Asian, Hispanic, and black women, which resulted in wide CIs. Furthermore, though we controlled for confounding by known risk factors of type 2 diabetes, we cannot completely exclude the possibility of residual confounding owing to the observational nature of the study. Generalizability of our findings may be limited because all the women in our study were health care professionals, and therefore they could be more health conscious than the general U.S. population. However, this would reduce the likelihood of confounding by socioeconomic variables that are hard to measure. Lastly, the FFQ designed for a population that consists mainly of NHWs may not fully capture ethnic foods and in turn may misclassify individuals with unusual or culture-specific diets. However, our FFQ has worked similarly in African American and European American groups (39), and the population of nurses in our cohorts may have been assimilated into U.S. culture by virtue of their education and employment. Moreover, misclassification of diet resulting from ethnic-specific dietary patterns would have tended to underestimate associations in minority groups.

In conclusion, our findings strongly support a protective association between a healthy diet and risk of type 2 diabetes in all racial and ethnic groups. We found that a healthier diet represented by a higher dietary diabetes risk reduction score was associated with lower risk of type 2 diabetes in both white and minority groups. The difference in absolute risks suggests that dietary improvements should confer even greater benefit for minority women than for NHW women with respect to type 2 diabetes risk. As the incidence of type 2 diabetes is increasing at an alarming rate worldwide, and will most likely continue to be a major cause of morbidity and mortality in coming years, it is crucial to understand the role of preventable lifestyle risk factors in the development of diabetes. Findings from this study suggest that healthy overall diet can play an important role in public health efforts to prevent type 2 diabetes, particularly in minority women, who have elevated risks of diabetes.

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Author Contributions. J.J.R. designed the research, performed the statistical analysis, drafted the manuscript, and read and approved the final manuscript. J.M., M.D.H., and F.B.H. contributed to the interpretation of data, made critical revisions to the manuscript for intellectual content, and read and approved the final manuscript. W.C.W. designed the research, contributed to the interpretation of the data, made critical revisions to the manuscript for intellectual content, and read and approved the final manuscript. J.J.R. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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